May the parathyroids be identified with the naked eye in the course of an operation? Yes, and almost infallibly; but perhaps not from any description that I can give. They are little ovoid, spheroid, lenticular, or very flat bodies, exhibiting much variety both in form and size and even in color. Externally they often resemble fat very closely in consistence as well as in color. On section, however, the fat about the deep cervical vessels and in the neighborhood of the thyroid gland is usually much whiter than the parathyroid, and the latter has a definite but exceedingly fine, almost microscopic, granular appearance. Differentiation from accessory thyroids or detached or partially detached lobulettes of normal thyroid tissue, is rarely difficult, although careful comparison of the suspected bodies with the tissue of the goitre may be necessary to convince one as to their nature. Partially or wholly detached portions of certain yellowish goitres, or adenomas of the thyroid, may bear a troublesome resemblance to the parathyroids. Lymphatic tissue is quite readily differentiated by the expert. Differentiation may be impossible if the parathyroids have been subjected to trauma, ever so slight.

The cause of tetany at the hands of well-versed operators is, I believe, less often removal of the parathyroids than interference with their circulation; and I am inclined to feel that the procedure of stripping the posterior capsule, so beautifully executed by Dr. C. H. Mayo, does not guarantee the safety of the parathyroid bodies. To assist in determining this point the removal of one lobe furnishes really no evidence whatever. It is only from the results of operations on both lobes that we can base an opinion. But who, in order to decide the question, will take the risk of causing tetany? I fear to do an operation, even on one side, except in very ill patients, which does not contemplate the leaving of a slice of the thyroid gland in the region of the ultimate distribution of the inferior thyroid artery. I avoid also ligation of the trunk of the superior artery, for fear of injuring the tiny branch to the superior parathyroid, and hence usually slice through the gland a little below the superior pole.

THE INTERPRETATION OF THE PULSATIONS IN THE JUGULAR VEINS.

By James MacKenzie, M.D.,
of Burnley, England.

Although the venous pulse has been recognized by clinicians for over two hundred years, the exact significance of its meaning today is not quite clear. It is only since graphic records have been taken of the movements in the veins that definite data have been obtained from which conclusions could be drawn. It was in 1863 that Bamberger and Marey published tracings of the venous pulse, being followed shortly by Geigel and Friedreich, but these tracings were taken independently of any timing with a standard movement. In 1868 Potain obtained tracings of the venous pulse simultaneously with the carotid artery, and after him numerous other observers, notably, Riegel, Francois-Franck, and Gerhardt investigated this subject. The general conclusion arrived at was that there were two kinds of venous pulse: one called the *normal* or *negative* venous pulse, the other the *pathological* or *positive* venous pulse. Generally speaking, this discrimination has prevailed in the literature of this subject.

In 1893,[1] I drew attention to the fact that the varieties of the venous pulse could not be satisfactorily included in these two simple forms, and that clinicians had entirely failed to appreciate the meaning and significance of the various forms of the venous pulse. The terms employed were utterly misleading: the so-called normal venous pulse was often as abnormal as the pathological venous pulse; the negative venous pulse was often largely composed of positive elements, and the positive venous pulse might often show negative evidence. As I had found that the presence of the auricular wave was a constant phenomenon in the majority of cases, I suggested that the term *auricular venous pulse* should be applied to these forms of pulsation in which the auricle occupied its normal place in the cardiac cycle, although in many cases evidence of the effects of the ventricular systole was present as well. On the other hand, I found that what had hitherto been called the positive venous pulse did not show any evidence of the auricular systole at its normal period in the cardiac cycle, and that the whole of the wave occupied the period of ventricular systole; I, therefore, employed the term *ventricular venous pulse*. Further, the appearance of the ventricular venous pulse implies a profound change in the rhythm of the heart, inasmuch as it is the evidence that the heart's contraction starts no longer at the mouth of the great veins.

VARIABILITY IN THE APPEARANCE OF THE VENOUS PULSE. To a great extent we are even at this day quite at a loss to explain all the conditions that give rise to a venous pulse. Some people in apparent good health show it, while in cases of marked heart failure it is entirely absent. Some people when in robust health show no signs of it, but if they become slightly debilitated the venous pulse may appear in the neck. In some cases of pernicious anemia the venous pulse may be a very prominent symptom, in others it may never appear. Some women during pregnancy develop a large venous pulse, others develop it only during the puerperium, while others never show the slightest sign of it. In irregular action of the heart it may assume, in certain cases, enormous proportions; in other cases there is not the slightest sign. A man may show it during one attack of heart failure, and during another and more grievous attack may show no sign of it. When one considers the foregoing variabilities in the appearance of the jugular pulse one is compelled to inquire more closely into the mechanism by which it is produced, and it will be found that our knowledge of the heart's action is even at the present day too slight to enable us to speak with certainty.

---

[1] The Venous and Liver Pulses, etc., Jour. Path. and Bact., 1894, ii. In this article a full bibliography is given up to 1893.

CARDIAC FACTORS CONCERNED IN THE PRODUCTION OF THE VENOUS PULSE. In order to appreciate fully all the factors concerned it is necessary that we should recognize the very valuable discoveries of Gaskell on the functions, and some observations of Keith on the structure, of the heart. Keith[2] has shown that the musculature of the heart is built up of bands of muscle fibers, having as distinct places of origin and insertion as skeletal muscles, and that each bundle has its function as peculiar to itself as a skeletal muscle. But the heart-muscle fibers are distinct from skeletal muscle in this respect, that according to their special duties certain portions have individual functions more developed than other portions.

The work of Gaskell[3] has shown that the contraction of the muscle fibers is due to the possession by these fibers of certain very definite functions: the functions of rhythmicity, excitability, conductivity, contractility, and tonicity. All the fibers of the heart are not equally endowed with the same functions, some fibers being naturally endowed with those functions which are essential to the playing of their part in the mechanism of the heart. Thus some fibers have the power of contractility more developed than others, for it is evident that the muscle fibers constituting the apex of the heart must have different functions to perform than those that support the auriculo-ventricular valves. In the latter case the function of tonicity is naturally that one that would subserve the purpose of these fibers. It follows, then, that if the muscle fibers that prevent the regurgitation of the blood from the right heart into the great veins have their tonicity affected, they cannot effectively assist in closing the caval and tricuspid orifices. Keith has shown that this regurgitation during auricular systole is prevented by the contraction of a band of muscle in the auricular wall (the tænia terminalis), which shuts off the auricle from the superior vena cava. In some of the hearts I have sent him he has found these fibers elongated and attenuated, and during life there was great regurgitation of the blood into the veins during the heart's contraction. In like manner the fibers that support the tricuspid valves are very apt to lose their tonicity, and as these valves even in health are barely sufficient to close the tricuspid orifice, the slightest depression of the tonicity of the muscle fibers at once permits regurgitation from ventricle to auricle.

---

[2] The Evolution and Action of Certain Muscular Structures of the Heart, The Lancet, February 27, 1904.

[3] The Contraction of Cardiac Muscle, in Schäfer's Text-book of Physiology.

TRICUSPID REGURGITATION. When I had taken tracings of the venous pulse and considered all the possible factors in its production, I took into consideration the fact that tricuspid regurgitation might play a very important part in the pulsation of the veins. Although its effects had been recognized in producing the so-called pathological venous pulse, no attempt had been made to show the connection between the so-called normal and the pathological venous pulses. That tricuspid regurgitation was the cause of the latter form was recognized, but no one had worked out how this tricuspid regurgitation began and gradually increased. Watching many cases and observing the signs of tricuspid regurgitation during life, and examining the hearts after death, I found abundant evidence that tricuspid regurgitation does modify even the so-called normal venous pulse. That my views were accepted by my colleagues in Britain was due to the fact that tricuspid incompetency is looked upon as so common, and so readily set up, that the whole train of my reasoning was conformable to their experience. But to my surprise I found that outside the British Isles my explanation was not accepted, as the fact that tricuspid incompetence was so easily set up had never been recognized.

Clinicians one hundred years ago, and John Hunter before them, recognized this tendency to incompetency of the tricuspid valves, but it was left to Wilkinson King,[4] in 1837, to demonstrate the relative natural incompetency of the tricuspid valves by a series of careful dissections and experiments. For several generations this has been the teaching in British schools, and in such text-books as those of Bramwell and Gibson the natural tendency to tricuspid incompetence is duly recognized. On the other hand, Babcock expresses surprise that Gibson should describe tricuspid regurgitation as a frequent occurrence, and, as an additional evidence of American opinion, Preble[5] recently bears testimony that tricuspid regurgitation is rarely recognized, and in citing some cases of pernicious anemia, he describes as new what has been taught in our schools for generations. Writers like Riegel, Krehl, Sahli, and others recognize tricuspid regurgitation when it is secondary to failure of the left heart, and in the extreme form when it produces the pathological or positive venous pulse. I have again carefully gone into the whole matter and made a series of new observations, noting the symptoms during life (particularly a murmur, systolic in time, heard loudest over the middle of the sternum, and over the third and fourth left costal cartilages), and I have followed certain patients until their death, and examined their hearts and found marked dilatation of the tricuspid orifice. But as even these cases might not be considered free from doubt, inasmuch as it could be held that the tricuspid dilatation was due to the changes taking place immediately preceding death, I have carefully noted the symptoms during life in patients whose tricuspid valves had been damaged by old-standing endocarditis. In these cases it would have been impossible for the valves to close efficiently, and regurgitation must have taken place. The results of my observations, as I will show, demonstrate that tricuspid regurgitation must have been a factor in producing the wave *v* in the auricular venous pulse in these cases.

---

[4] The Safety-valve Function in the Right Ventricle of the Human Heart, Guy's Hospital Reports, 1837, ii, 132.

[5] AMER. JOUR. MED. SCI., 1906, cxxxii, 393.

METHODS OF RECORDING THE MOVEMENTS OF THE CIRCULATION. The examination is best carried out with the patient lying down, with the shoulders slightly raised, and the head comfortably supported by a pillow, so that the sternomastoid muscle can easily be relaxed. A great variety of movements may be observed in different patients.

There are several ways of taking tracings of the movements in the jugular vein. Usually the movements are recorded on the blackened paper of a revolving drum or of a sphygmograph at the same time as some other standard movement, such as the apex beat or arterial pulse. These methods, besides being troublesome, permit only of tracings being taken for a short period, and it is often necessary in the study of irregularities to take tracings extending over a considerable time. I have lately devised a method whereby the lever of the instrument writes, with ink, on paper that is unwound from a roll, so that tracings many yards in length can be taken with much less trouble. For taking movements of the jugular and the carotid pulses or of the apex beat I employ a shallow cup (receiver). When this is applied over the pulsating part in such a manner that all communication with the outer air is prevented, the movement of the air within the receiver is conveyed by an India-rubber tube to the tambour. I have devised a special arrangement for taking a tracing of the radial pulse: the knob on the membrane of a broad tambour rests on a steel spring which is moved by the radial pulse, the steel spring being in a splint which is strapped to the wrist. A time marker, driven by a separate clockwork, records one-fifth of a second. Most of the tracings for this paper have been taken by this method.[6]

For the due appreciation of the movements in the jugular vein it is absolutely necessary that, along with the jugular pulse, the movements of some event whose position in the cardiac cycle is known, be taken at the same time as a standard, by which to determine the position in the cardiac cycle of any movement in the jugular pulse. For this purpose the arterial pulse usually offers the most convenient movement. Its cause—ventricular systole—is absolutely determined. Fig. 1 shows the movements which serve as standards for the interpretation of a jugular pulse. I have placed below the jugular pulse, movements representing the apex beat and the carotid and radial pulses to show the relation in time of certain events in these various movements. The numbered down strokes indicate the time relations of the events in the jugular pulse, the apex beat, the carotid and the radial pulses: (1) The beginning of auricular systole; (2) the beginning of ventricular systole; (3) the opening of the semilunar valves and the appearance of the carotid pulse; (4) the beginning of the radial pulse; (5) the closing of the semilunar valves; and (6) the opening of the tricuspid valves. The space *E* between 3 and 5 indicates the time when the semilunar valves are open. These numbered down strokes and the space *E* have the same significance in all the other tracings. The time marked here and in all the other tracings equals one-fifth of a second.

---

[6] This instrument is made by Mr. S. Shaw, watchmaker, Padiham, Lancashire, England.

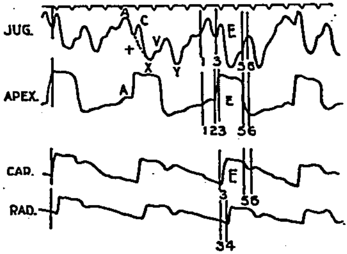

FIG. 1.—Tracings of the radial, carotid, and jugular pulses and of the apex beat.

Although the carotid pulse and the apex beat can sometimes be usefully employed as a standard, it will be found that the radial pulse is, on the whole, the most convenient. A certain loss of time takes place in the transmission of the wave to the radial pulse, and this can be estimated by taking simultaneously with the radial a few beats of the carotid, as in Figs. 2 and 10, in which the space between 3 and 4 shows the loss of time between the appearance of the carotid and the radial pulses. This loss being allowed for, one can always find a definite period in a jugular tracing which corresponds to any event occurring in the neck due to the ventricular systole. I also employ a very useful period, namely, that portion of the ventricular systole during which the semilunar valves are open (period marked *E* in all the tracings). Its duration can be found in the radial tracing, extending from the beginning of the up stroke to near the bottom of the dicrotic notch. It corresponds to the time between the down strokes 3 and 5 in all the carotid, jugular, and apex tracings given here. In the neck the period *E* begins with the carotid pulse.

There is a slight delay between the opening of the aortic valves and the carotid pulse, but it is so short ($\frac{1}{50}$ of a second) that it may be ignored. The space *E* in the radial tracing is about one-tenth of a second behind the same period in the neck.

Another standard which I will be able to point out later is that of the opening of the tricuspid valves (downstroke 6 in all the tracings), which in jugular tracings is always indicated by the beginning of the fall of the wave *v*. In the apex tracing this event occurs at the bottom of the fall after the systolic plateau as in Figs. 22 and 23.

INTERPRETATION OF A JUGULAR TRACING. 1. *The Auricular Wave* (*a*). To interpret such a tracing as Fig. 2 we find the space *E* (that is the period during which the semilunar valves are open). There is a wave *a* immediately preceding this period. From its position in the cardiac cycle this wave can only have been due to the auricular systole. Although it is recognized as being due to the systole of the auricle, there is some doubt as to its manner of production.

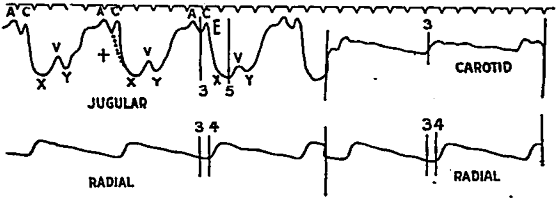

FIG 2.—Simultaneous tracings of the jugular and the radial pulses in the first part of the tracing and of the carotid and the radial in the latter part. The jugular pulse is of the auricular type. *a*, auricular wave; *c*, the carotid wave; *v*, the ventricular wave; *x*, the auricular depression; *y*, the ventricular depression. These letters have the same significance in all the other tracings.

Thus, it has been said to be due: (1) To a damming of the blood in the vein while the auricle is in systole; (2) to the fact that when an on-flowing stream is suddenly stopped there immediately takes place a wave in a contrary direction; and (3) to a true regurgitant wave. This is really a matter of some moment, as a due appreciation of this wave helps materially in understanding certain movements of the heart. Keith, as I have remarked, has described that regurgitation is prevented by the closure of the superior vena cava by the muscular band, the tænia terminalis. If the tonicity of this bundle be affected it will lengthen and become less effective. I am disposed to consider that it becomes readily affected by distention of the auricle under certain circumstances, even if it is ever really competent. In some of my cases in which there was a marked venous pulse, Keith has found this bundle greatly lengthened and atrophied, and I would therefore suggest that this wave is a true regurgitation wave and that it is due to inefficiency of the contraction of the tænia terminalis. I may point out in support of this view that Chauveau and Faivre[7] found in the horse that normally there is a slight regurgitation during auricular systole, which becomes markedly increased when the breathing is stopped.

2. *The Auricular Depression* (x). After the auricular systole there is a fall (x in all the tracings). It is usually the greatest depression in a tracing of the auricular form of venous pulse. If we leave out, for the moment, the consideration of the wave c, and consider the fall as following the dotted line at + in Figs. 1, 2, and 22, this fall will be found to occupy the whole period of E in many of the tracings, while in others it stops at varying periods before the end of E (Figs. 10, 12, 14, etc.). This fall is due to three factors, of which the first and principal is the relaxation of the auricle after its contraction, whereby the blood in the vein is suddenly passed into the dilating auricle. As a rule, it cannot be said how much is due to this factor and how much to the other factors, but in exceptional instances, in which we get a delay in the appearance of the ventricular systole, we find this fall divided in two, as (x and x^1) in Fig. 13.

After the auricular systole in this tracing, there is a slight delay before the wave c appears, and the fall x, between a and c, is due to the auricular diastole. The period E marks the time of ventricular systole, and we find, during this period, that there is another fall x^1. The cause of x^1 is, as was first pointed out by John Hunter, the diminution of the intrathoracic pressure, caused by the expulsion out of the chest of the blood by ventricular systole. It is this diminished intrathoracic pressure, which causes the aspiration of the air in the respiratory tract, which has been studied by Mosso, Landois, François-Franck, Delépine, and others. There is no doubt that it must also have some effect on the flow of blood in the veins and in producing the fall x^1 in Fig. 13.

Keith has pointed out to me that another factor would be the enlargement of the auricular cavity by the dragging down of the auricular-ventricular septum during ventricular systole. Chauveau had previously described a similar action, and probably it is a factor in the production of this fall. In a tracing from a case of heart block Wenckebach[8] has also demonstrated a division of this fall and gives an explanation similar to this.

3. *The Carotid Wave* (c). The carotid wave c gave rise to much discussion in the earlier days, but I was able to demonstrate, in 1893, that it is really due to the impact of the carotid on the air in the receiver, and is not due to a wave in the vein. I was able to produce such ample evidence that this interpretation was generally

---

[7] Nouvelles recherches expérimentales sur les mouvements et les bruits normaux du cœur. Gazette médicale de Paris, 1856, v, 406.

[8] Beiträge zur Kentniss der menschlichen Herztätigkeit, Archiv f. Anatomie und Physiologie, 1906, 297.

accepted, but I find that Hirschfelder[9] and Morrow[10] have recently revived the old notion that it is in some way due to a wave sent back by the ventricle into the jugular vein.

It can be proved in many ways to be due to the carotid. Thus its synchronism with the carotid pulse is seen in Fig. 3. Clinically, we can demonstrate the nature of this wave by taking tracings from the neck at various levels. As we ascend higher in the neck the jugular element in the tracing will gradually disappear, while the carotid element will increase until nothing but the characteristic carotid pulse is left in the tracing. I have demonstrated this fact in many other ways, as, for instance, its absence when the jugular wave is large, as in Fig. 8, and its absence from the auricular liver pulse, as in Fig. 9. Morrow reckons to have demonstrated experimentally that this wave is due to the ventricular systole, but I am disposed to think that his experiments are not reliable. At all events, when able experimenters like Cushny and Hering took pure tracings of the jugular pulse they were able to get a tracing without a carotid wave.

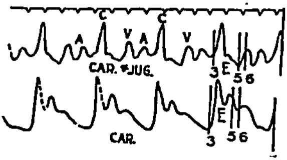

FIG. 3.—Simultaneous tracing of the jugular and the carotid pulses to show the synchronism of the wave c in the jugular with the carotid pulse.

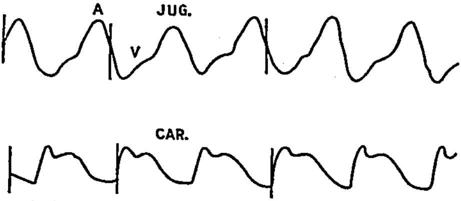

FIG. 4.—Simultaneous tracings of the jugular and the carotid pulses from a dog when the jugular had been dissected away from the carotid, showing the absence of a carotid wave in the jugular tracing (Cushny and Grosh).

Fig. 4 shows a jugular tracing from a dog taken by Cushny and Grosh after they had dissected the jugular vein away from the carotid.

---

[9] Graphic Methods in the Study of Cardiac Disease, AMER. JOUR. MED. SCI., 1906, cxxxii, 378.

[10] Discussion on Heart Block, Brit. Med. Jour., October 27, 1906, and The Various Forms of the Negative or Physiological Venous Pulse, Brit. Med. Jour., December 22, 1906.

It will be seen that there is no wave in the jugular tracing at the time of the ventricular systole, although in tracings taken prior to this dissection the carotid wave was always present. Likewise, Hering's[11] tracings, after he had detached the vein from the carotid, showed no sign of a wave at this time.

4. *The Ventricular Wave* (*v*). The next event to be considered is the wave marked *v* in all these tracings. It will be observed that I mark with *v* the small wave occupying a very short period in the cardiac cycle, in Figs. 1 and 22, as well as the large wave in Fig. 23, for the reason that the smaller wave is but the beginning of the larger. The best way to study this wave is to begin at its end. What causes the fall that terminates it? If careful measurements be taken to find in what portion of the cardiac cycle the fall occurs, the beginning of the fall will be found to occur at the time of the opening of the tricuspid valves (6 in all the figures). As a matter of fact, the fall of this wave is due to the opening of the tricuspid valves, permitting the blood that has been stored in the auricle during ventricular systole to flow into the ventricle, thus emptying the jugular vein. If this fact be kept carefully in mind, the real significance of the wave *v* becomes clear; its cessation marks a very definite period in the cardiac cycle and forms a valuable standard in the interpretation of many obscure tracings. The manner of origin of this wave is as follows: While the pressure in the ventricle during ventricular systole is higher than that in the auricle, the tricuspid valves will remain closed. The flow of blood into the auricle will continue to accumulate until the auricle is filled and the overplus will be stored in the venæ cavæ and adjacent veins. When the overplus is sufficiently great it will appear in the jugular as a wave. If the overplus is slight the capacious vena cava may retain it all, so that it will not appear in the jugular before the tricuspid valves are opened. The greater the overfilling of the auricle and the veins the earlier will be the appearance of the wave in the period *E* of ventricular systole.

The question is, Whence comes the blood that produces this filling and overfilling of the auricle? In the majority of people the jugular pulse is only apparent when the individual is recumbent. This posture favors the flow to the heart from the abdominal veins, and in certain circumstances this seems readily to lead to overfilling of the right heart. Another source to be considered is the regurgitation back from the right ventricle. I have examined very carefully the state of the heart in several thousands of patients who had a jugular pulse, and I have found that in many of them I could detect a tricuspid systolic murmur and slight dilatation of the right heart. If regurgitation does take place, in what way will it affect the jugular pulse? If the amount be slight it will only cause an earlier filling

[11] Die Verzeichnung des Venenpulses am isolierten, kunstlich durchströmten Säugethierherzen, Archiv f. die ges. Physiologie, 1904, cvi.

of the auricle, and this may be only sufficient to cause the overfilling to extend as far as the jugular, causing a wave to appear just before the opening of the tricuspid valves between the down strokes 5 and 6, as in Figs. 5 and 22. If, on the other hand, the regurgitation be more free the filling of the auricle will occur sooner and the wave appear earlier in the jugular tracing. This is, as a matter of fact, what I have found to take place.

Fig. 5 is from a man suffering from shortness of breath and tightness across the chest, in consequence of a very laborious occupation. There was slight enlargement of the heart and a well-marked tricuspid systolic murmur. Fig. 6 is from a profoundly anemic woman, with a large heart and a loud tricuspid systolic murmur, in whom there was a large pulsation in the veins of the neck. I have followed patients similar to these until they died, and found postmortem evidences of great dilatation of the tricuspid orifice.

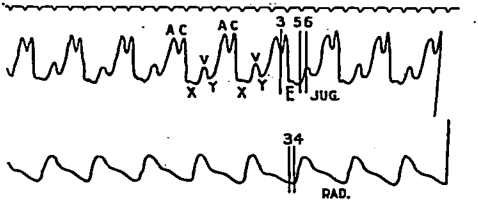

Fig. 5.—Simultaneous tracings of the jugular and the radial pulses from a man with tricuspid regurgitation. The jugular pulse is of the auricular type and shows a small wave after the period *E*.

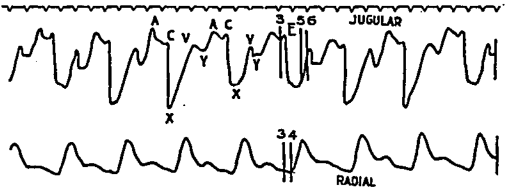

Fig. 6.—Simultaneous tracings of the jugular and the radial pulses. The jugular pulse is of the auricular type. From a profoundly anemic woman with marked tricuspid regurgitation.

Thus, Fig. 7 is from a man who suffered from pernicious anemia. There was slight enlargement of the heart and a tricuspid systolic murmur. He died, and on postmortem examination the tricuspid orifice readily permitted the passage of four fingers. In such cases I suggest that the depressed tonicity of the muscular ring supporting the tricuspid valves permitted enlargement of the orifice and consequent incompetence of the tricuspid valves.

As some doubt may even here exist in regard to the diagnosis, I would call attention to instances of undoubted tricuspid regurgitation in patients in whom the tricuspid valve had been partially destroyed as a result of old endocarditis, and in which there could be no doubt of the fact that tricuspid regurgitation had occurred during life. I have had postmortem examinations on 8 cases of moderate tricuspid stenosis in which the opening permitted the passage of one or two fingers; in 6 of these there was pulsation of the auricular form in the jugular vein and the liver, and I had taken a large number of tracings of these pulsations.

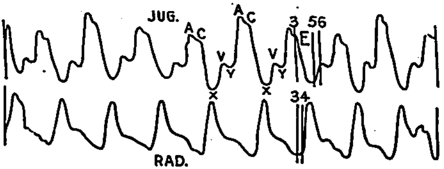

Fig. 7.—Simultaneous tracings of the jugular and the radial pulses from a man suffering from pernicious anemia. At the postmortem examination the tricuspid orifice was widely dilated. The jugular pulse is of the auricular type.

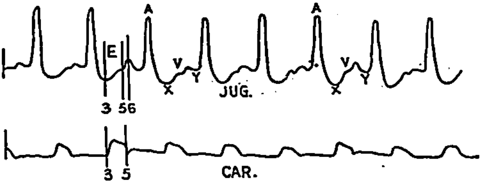

Fig. 8.—Simultaneous tracing of the jugular and the carotid pulses from a patient in whom the tricuspid valves were found damaged by an old endocarditic process, and the tricuspid orifice thereby rendered markedly incompetent. The jugular pulse is of the auricular type.

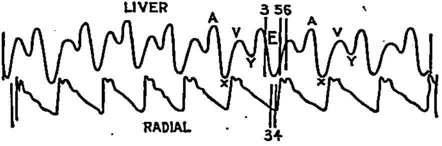

Fig. 9.—Simultaneous tracing of the liver pulse and the radial pulse from another patient whose tricuspid valve had been rendered incompetent because of an old endocarditis. The liver pulse is of the auricular type.

Fig. 8 is a tracing of the jugular pulse from one case, and Fig. 9

is a tracing of the liver pulse from another. In an analysis of these tracings it will be found that the wave *v* is small and appears only toward the end of ventricular systole, and its fall begins at the time of the opening of the tricuspid valves. That is to say, in the people from whom these tracings were taken, tricuspid regurgitation undoubtedly occurred, and in doing so it must have modified the wave *v* in the tracings. I cannot conceive how it could affect these tracings in any way other than in participating in the earlier filling of the auricles and veins and, therefore, in the production of the wave *v*.

I have selected purposely, to illustrate my argument, cases in which there could be no reasonable doubt as to the occurrence of tricuspid regurgitation, in order to impress the fact that this factor must be concerned in the production of the wave *v*. As I have said, I have taken tracings of several thousand cases and have studied at the same time the condition of the heart. In the large majority of cases no tricuspid murmur could be heard, and in many cases there was practically no increase in the size of the heart.

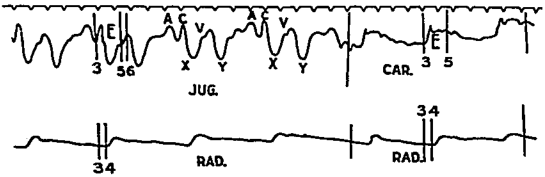

Fig. 10.—Simultaneous tracing of the jugular and the radial pulses in the first part of the tracing, and of the carotid and the radial pulses in the latter part. From a man without a tricuspid systolic murmur.

Thus, Fig. 10 was taken from a pale, spare man, aged twenty-five years, with dilated stomach. There was no cardiac enlargement and no systolic murmur. He was rather short-winded. The wave *v* encroaches on the period *E*. What is the explanation of such cases? Tricuspid regurgitation is not excluded by the absence of a systolic murmur. One can frequently demonstrate the existence of large tricuspid regurgitation, as shown by a ventricular venous pulse, with the absence of a tricuspid regurgitant murmur. Careful clinicians are well aware that under certain circumstances regurgitation can occur at all the orifices with no murmur. Thus, for instance, in the enormously dilated hearts of adherent mediastinitis there may be no systolic murmur though the mitral and tricuspid orifices are greatly widened. The question, therefore, whether tricuspid regurgitation takes place in such cases as the man from whom Fig. 10 was taken is one on which I have not sufficient evidence to decide. As I have already pointed out, the essential element in the production of the wave *v* is a sufficient accumulation of blood in the right auricle, so that the auricle and superior vena cava are overfilled during the period of the cardiac cycle when the tricuspid valves are closed. This can occur with a slight engorgement of the right heart, when there is an increase in the residual blood after the auricular systole. The jugular pulse in these cases readily disappears with more rapid action of the heart, as on moderate exertion, or on the supervention of a slight febrile attack, and often increases in size with a slower action of the heart. It is probable the mere increase of the accumulated blood in the right heart may suffice alone to produce the wave *v* in such cases, but whether it may at the same time render the tricuspid valve incompetent is a question that must be considered, but which I am unable to answer.

Before demonstrating how the auricular venous pulse passes into the ventricular, I will consider some of the objections raised against the foregoing explanation. The erroneous notion very commonly raised by people who have not fully considered all the factors concerned and found in text-books, physiological and clinical, is that if there were tricuspid regurgitation in such cases as in the foregoing instances the wave *v* ought to appear at the beginning of ventricular systole and not at the end. If the jugular vein was in such direct communication with the right ventricle as the carotid is with the left, then undoubtedly the wave *v* should appear at the same time as the carotid. *But it must be borne in mind that between the right ventricle and the jugular vein there is interposed a dilating auricle and a capacious superior vena cava, and that a wave can only appear in the jugular after the auricle and vena cava have been overfilled.* It is for this reason that the time of the appearance of the wave *v* in the period *E* is a measure of the engorgement of the right auricle, as I shall demonstrate shortly.

It has been suggested by Gerhardt, Wenckebach, and others that the wave *v* in such tracings as Figs. 2 and 5 is due to a backward movement of the auriculo-ventricular septum after the closure of the semilunar valves. They state that at this period, when relaxation of the ventricle takes place, the septum swings back, sending a wave through the auricle and superior vena cava into the jugular. I am too ignorant of the movements of the heart to say whether there is such a movement at this time, and the arguments given in favor of it are more presumptive than demonstrative. Admitting, however, that it does occur, it would only be a factor added to those I have already given, and would not account for the portion of the wave occurring during the period *E* in such tracings as Fig. 10. I called attention, in 1893, to the fact that there is often a notch in the jugular tracing at the time of the closure of the semilunar valves, but its cause is still obscure. If this return of the auriculoventricular septum does take place it cannot be of such great force, as, for instance, to distend the liver and raise one's hand when laid on the liver, for one can see the hand raised by this wave, in palpating the liver in cases of auricular pulsation of that organ. Another curious feature is that those who support this view leave out of consideration altogether the manner in which this wave *v* grows forward into the period *E.* Lately two physiologists, Hering and Morrow, by experiments on dogs, have sought to show that this wave is a normal sign and has nothing to do with tricuspid regurgitation. Their experiments are not conclusive, for they do not seem to have tested whether the tricuspid valves were competent, nor did they seek to produce tricuspid incompetence and observe the result on such incompetence. I do not see how they can say that tricuspid incompetence did not participate in the production of the waves in the cases cited here when the tricuspid valves were damaged.

5. *The Ventricular Depression* (*y*). The next event after the wave *v* to be seen in the jugular and liver pulsation is the fall marked *y* in all these tracings. Its cause is readily understood, being due to the passage into the ventricle of the blood stored in the auricle during ventricular systole. It will be noticed that in all these tracings the fall begins at the down stroke 6, that is at the time of the opening of the tricuspid valves. If the heart be slow in rate it is followed by a rise shown in Fig. 2, between *y* and *a*. This rise is due to stasis in the veins. If the heart quickens in its action the period of stasis first disappears and the fall *y* is followed at once by the auricular wave. If further quickening occurs the fall *y* disappears from the tracing, and the wave *v* becomes continuous with the auricular wave, as in Fig. 11.

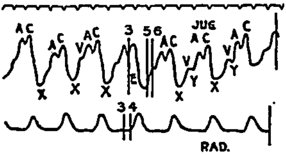

FIG. 11.—To show the disappearance of the fall *y* and the wave *v* with quickening of the heart rate.

In my book on the pulse I have given numerous illustrations of these features, so that I need not dwell on them here. As a rule, the fall *x* is more marked than the fall *y*, in the auricular form of the venous pulse; but, as the pulsation passes into the ventricular form the fall *y* gradually becomes the more pronounced, and the fall *x* diminishes, until it disappears altogether, until there is but one large wave synchronous with and due to the ventricular systole, and one great fall *y*, synchronous with and due to the ventricular diastole. The manner in which this occurs I will now proceed to demonstrate.

THE PASSAGE OF THE AURICULAR FORM OF THE VENOUS PULSE INTO THE VENTRICULAR. The reason I called the wave marked *v* the *ventricular wave* in the foregoing tracings, although it may be that the ventricular systole in many instances takes no part in its production by sending blood back into the auricle, was to impress the fact that had been overlooked, that tricuspid regurgitation must have had a beginning, and that the "positive" or ventricular jugular pulse, which everyone recognizes as due to extreme tricuspid regurgitation, began its appearance as a small wave appearing at the end of ventricular systole. How necessary it was to insist on this fact is seen in the writings of those who have not recognized early tricuspid regurgitation. Scarcely a writer ever attempts to explain how the "positive" venous pulse comes about. The reason for this silence is, that these writers, clinicians as well as physiologists, have failed to recognize the important factor of slight and moderate tricuspid regurgitation. I quite admit that to follow my argument it may require a clearer exposition than I am capable of giving, but my facts cannot be gainsaid. If, therefore, those interested in this matter will ponder deeply on this somewhat complex problem, I am convinced they will see the explanation as clearly as I do.

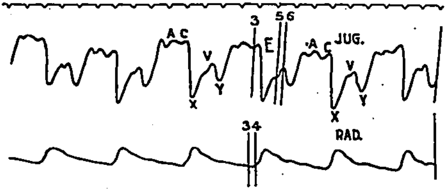

FIG. 12.—Shows the extension forward of the wave *v* into the period *E* when the patient held his breath. Compare with Fig. 5, taken from the same patient.

While the following explanation shows how the auricular form of the venous pulse passes into the ventricular, it affords, at the same time, proof of varying degrees of engorgement of the right heart and demonstrates the manner in which an increasing engorgement can be recognized in tracings of the jugular pulse. I am somewhat at a loss in selecting illustrations, as no two cases are alike in all particulars, and I have by me such an abundance of material illustrating the view I am propounding, that in the limits of this paper I am restricted to giving only a very small portion of my proofs. In suitable cases it is possible to increase the amount of right-heart engorgement by getting the patient to hold his breath. In many instances in which the jugular pulse is of the ventricular type, it may occasionally disappear, but can be brought back by holding the breath. In some cases in which the jugular pulse is of the auricular form the wave *v* can be made to increase in size and to appear earlier in the cardiac cycle by the same method. Thus, Fig. 5 shows *v* appearing only after the period *E*. When I told this patient to hold his breath for a long time the jugular pulse increased in size and the wave-*v* extended forward into the period *E*, as in Fig. 12.

Here I reason that the greater resistance offered to the systole of the right ventricle by the cessation of the breathing had caused a rise in the ventricular pressure and an increased backward flow. This explanation corresponds with that of Hering's[12] experiment when he found he had difficulty in raising the pressure in the right ventricle, because of the ease with which the tricuspid leaked.

The best evidence of increasing engorgement is to be found by watching individual cases for many years and noting the gradual advance of the cardiac inefficiency. The following case is a good illustration of this method: I attended a man during an attack of acute endocarditis, in 1881, he then being thirty-two years of age. He was left with a mitral regurgitant murmur. In my notes of his case I find a tracing of his jugular pulse taken in 1892 (Fig. 13), which shows that the wave *v* began a little before the end of the period *E*. At that time he was in fair health. It will be noted in the tracings from this patient that the carotid wave *c* follows the wave *a* at a greater interval than in the other tracings of the jugular pulse given in this paper. This delay implies a depression of conductivity of the fibers joining auricle and ventricle. In some tracings,

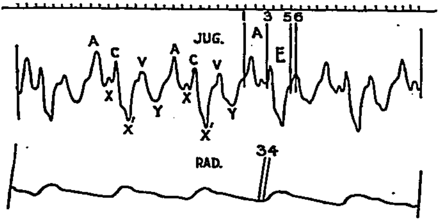

FIG. 13.—Note the size of the wave *v* in this and the following tracings. W. H., 1892.

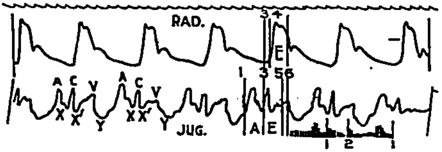

FIG. 14.—The shading underneath the jugular tracing shows the position of the heart murmurs. 1 and 2 refer to the first and second sounds. Note the increase of the diastolic murmur at the time of the auricular systole. W. H., 1903.

---

[12] Zur experimentellen Analyse der Unregelmässigkeiten des Herzschlages. Archiv f. die ges. Physiologie, Bonn, 1900, lxxxii.

taken in 1897, I find this had increased so much that there was a slight heart block. In 1898 he suffered from an attack of grievous heart failure, from which his recovery was slow and protracted. In 1903, I took the tracing, Fig. 14. Here it will be noted that the wave *v* appears still earlier in the period *E*. At this time there was, in addition to the mitral systolic murmur, a long diastolic murmur with a curious increase of intensity a little before the first sound, a brief interval occurring between this increase and the first sound, corresponding to the shading underneath the jugular tracing. It will be seen that the rise in the shading occurs exactly at the time of the auricular systole. It was my custom to ask my colleagues when showing them this patient, to draw for me on a tracing the period in the cardiac cycle in which this increase in the intensity of the diastolic murmur occurred, and in every instance they placed the period of increase exactly where I have shown it, although they could form at the time no opinion as to the cause of the increase. There can be no doubt that the increase in the loudness of the murmur at this time was due to the auricular systole.

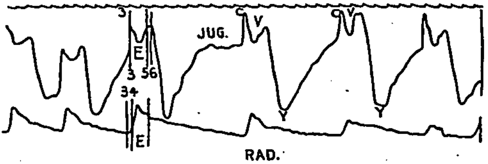

FIG. 15.—Simultaneous tracing of the jugular and the radial pulses. Note the irregularity and the absence of an auricular wave as compared with Figs. 13 and 14. The jugular pulse is now of the ventricular type. W. H., April 6, 1904.

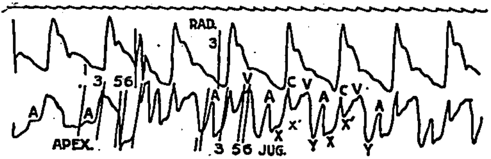

FIG. 16.—Simultaneous tracings of the radial pulse and the apex beat in the first part of the tracing and of the radial and the jugular pulses in the latter part. The pulse is again regular and the auricular wave *a* has returned to the apex beat and the jugular pulse. W. H., April 11, 1904.

The character of the jugular pulse on repeated examinations continued like Fig. 14 until April 6, 1904, when I found the pulse continuously irregular, and on listening to the heart there was a systolic and a diastolic murmur at the apex, but no increase in the diastolic murmur before the first sound. On taking a tracing all sign of the auricular wave had gone from the jugular pulse and there was but one large wave during the ventricular systole (Fig. 15). A week later the pulse was found to be quite regular and the jugular tracing showed the auricular wave had returned (Fig. 16). At the beginning of this tracing there are a few beats from the apex which show a wave, due to the auricle. I have taken a large number of apex tracings and never failed to find this wave when the rhythm was regular. The heart continued quite regular until the month of August, 1904, when I found it again continuously irregular and it has remained thus ever since.

Figs. 17 and 18 were taken on December 9, 1906, and are typical of the form of arrhythmia that I have found in the numerous tracings I have taken during the last two years—one large wave synchronous with the ventricular systole, no sign of an auricular wave in the tracing of the apex beat or the jugular pulse, no murmur due to the auricular systole, and invariably irregular. To appreciate more clearly the changes in the jugular pulse I arrange one cardiac cycle from Figs. 13, 14, 16, and 17, above one another, showing thus in a semi-diagrammatic form the gradual increase of the wave *v*, and bringing out clearly its meaning (Fig. 19). This demonstration of the passage of the auricular form of the venous pulse into the ventricular justifies the names I have given them, for it is seen in these tracings that the disappearance of the auricular wave marks at once a remarkable

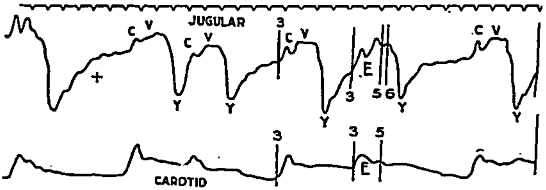

FIG. 17.—Simultaneous tracings of the jugular and the carotid pulses. The jugular pulse is now of the ventricular type. Note the irregularity and the small ripples in the tracing at the long pause +, due in all likelihood to fibrillary contraction of the auricle.

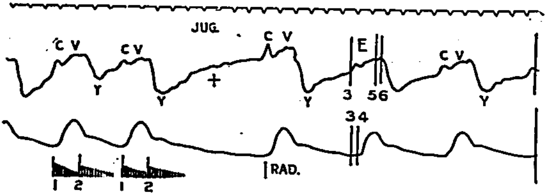

FIG. 18.—Here the jugular pulse is taken with the radial pulse and shows the same features as Fig. 17. Note the shading showing the murmurs and sounds of the heart, and compare with Fig. 14. W. H., 1906.

change not only in the character of the venous pulse but also in the rhythm of the heart. I wish to insist upon these facts, because though much has been written on mitral stenosis the clinical signs have never been properly appreciated, not even the fact that when the heart becomes continuously irregular, as it often does in advanced mitral stenosis, a presystolic murmur of the crescendo type is never heard. A diastolic murmur may be present and it may last during the whole of a short diastole, but if care be taken it will be found never to terminate in a crescendo murmur, and when the diastole is prolonged the murmur will be found to stop short of the first sound, as shown in the shading of Fig. 21.

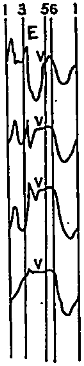

Fig. 19

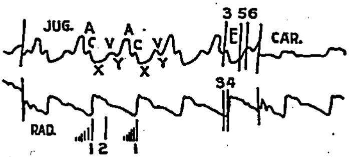

Fig. 20

Fig. 19.—Semidiagrammatic illustration of the passage of the auricular form of the venous pulse into the ventricular. Note the gradual increase in the size of the wave v.

Fig. 20 is from a case of mitral stenosis, taken in 1896, and shows a regular heart and a jugular pulse of the auricular form. The crescendo murmur is represented in the shading running up to the first sound. Mrs. P., 1896.

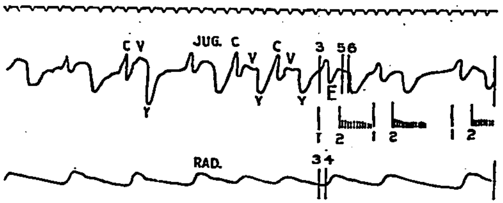

Fig. 21.—From the same patient as gave Fig. 20, taken in November, 1906. The rhythm is now irregular and the jugular pulse is of the ventricular type. The shading shows a diastolic murmur and an absence of the crescendo character represented in Fig. 20. Mrs. P., 1906.

Fig. 20 is a tracing taken in 1896, from a woman with mitral stenosis. The pulse is regular and the jugular shows the usual form of auricular venous pulse. The shading shows the position of the crescendo presystolic murmur running up to the first sound. In 1900 her heart became irregular and has remained so ever since. Fig. 21 is a tracing taken in 1906, and is characteristic of the irregularity and type of venous pulse—the ventricular—that has been present the past six years.

When the patient's heart beats quickly, as after exertion or excitement, the rate being rapid, the rhythm is fairly regular and a diastolic murmur fills up the whole of the diastolic period and might be mistaken for a presystolic murmur. But there is no crescendo character present in it. When, however, the heart becomes quieter, the irregularity becomes more marked, and long diastolic pauses ensue, and then it will be found that the diastolic murmur does not fill up the whole diastolic interval but stops short, as shown in the shading of Fig. 21. I have been able to demonstrate this peculiarity in a great number of cases to many of my colleagues. The study of the jugular tracings at once reveals the reasons for these changes. Progressive narrowing gives rise to, first, a crescendo presystolic murmur, then there is added a diastolic murmur, then the auricle ceases to beat at its normal period of the cardiac cycle and the crescendo murmur disappears, and the heart becomes irregular. In advancing mitral stenosis, therefore, these events happen simultaneously: (1) Disappearance of the auricular wave from the jugular pulse and apex beat; (2) disappearance of the auricular systolic (crescendo presystolic) murmur; and (3) arrhythmia of the heart. So far as the venous pulse is concerned this means the change from the auricular form to the ventricular.

It so happened that my earlier cases of the ventricular form of the venous and the liver pulses were all secondary to disease of the mitral valve, and at the postmortem examinations I had found the right auricle enormously distended and the muscle fibers greatly atrophied, so that I surmised that the auricle, like an overdistended bladder, had become paralyzed. This view seemed to be confirmed by the results of careful observation of individual patients extending over many years, the auricle never putting in an appearance at its normal period in the hundreds of tracings I have taken. This view I expressed in my book, but when writing it a few cases came under my observation that gave me grounds for suspecting that "auricular paralysis" was not the only condition that gave rise to the ventricular form of the venous pulse.

I, therefore, started on an inquiry into all forms of arrhythmia, and collected a large number of cases. From among these I was able to differentiate a group of over 500 cases in which the irregularity was of a disorderly nature, as in Figs. 21 and 23. Over one-half of these cases had a venous pulse, and in every case it was of the ventricular form. In more than 50 cases the disorderly rhythm had started when the patient was under observation, and it invariably meant the change from the auricular form to the ventricular—often suddenly. Usually, these cases for years have an occasional irregularity due to ventricular extrasystoles, that is, when the ventricle contracts before the auricle and independently of the stimulus from the auricle. I shall cite only one case as an illustration, particularly, as it has some historical value, being the first human subject in whom I demonstrated the nature of ventricular extra-systole.

In 1892, I took the tracing Fig. 22. In many tracings taken from this patient up to 1906 the venous pulse was always of the auricular form, as in this tracing, though she frequently showed ventricular extrasystoles, which are given in my book (Figs. 69 and 279). I saw her on August 6, 1906. The pulse had suddenly become quick and irregular and the jugular pulse was of the ventricular type. By November it had slowed down, and Fig. 23 is a characteristic tracing of its present condition.

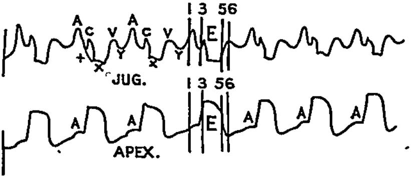

FIG. 22.—Simultaneous tracings of the jugular pulse and the apex beat. The jugular pulse is of the auricular type. Compare with Fig. 23. Mrs. A., 1892.

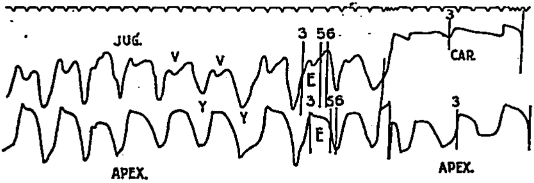

FIG. 23.—Simultaneous tracings of the jugular pulse and the apex beat. The jugular pulse is now of the ventricular type. Mrs. A., November, 1906.

Compare here the disorderly rhythm, the absence of the auricular wave in the jugular, and the apex tracing, with the perfect regularity and the presence of the auricular wave in the jugular and apex tracing of Fig. 22. Note, however, the persistence of the wave *v* between the down strokes 5 and 6 in both cases.

What is the auricle doing in cases with the ventricular venous pulse? I had assumed that it was paralyzed, and the total absence of any sign of its action for years, and the extreme distention of the chamber and its atrophied walls seemed to support the view. In some cases I have found evidence that it contracts at the same time as the ventricle, and on postmortem examination these cases did not show extreme atrophy of the auricular muscle. In a few other cases I sometimes found a series of small waves on the ascending line of the tracing preceding the ventricular wave, as at + in Figs. 17 and 18. The waves are sometimes larger than those in these tracings, as at + in Fig. 24, which was taken from the same patient that gave Fig. 22 and 23.

In discussing this matter with Professor Cushny he said he had no hesitation in accepting the view that these small movements are due to fibrillary contraction of the auricle, as he had frequently witnessed the auricle in fibrillary contraction while the ventricle went at a slower rate.

One conclusion I have come to, and it is this, that when the ventricular venous pulse is present the heart's contraction proceeds no longer from the normal place, that is, at the mouths of the great veins, but probably at the fibers joining the auricle and the ventricle. At present, Dr. Keith is conducting a minute inquiry into the condition of these fibers in a number of hearts I sent him of patients from whom, during life, I have taken numerous tracings, and so far it would seem that he will be able to show some anatomical facts justifying the foregoing conclusion. If this is so it will throw a flood of light on many obscure cases of heart failure which are at present buried under such vague terms as "chronic myocarditis," "fatty heart," "mitral irregularity," etc. It opens up a new view to clinicians and physiologists, and its bearing on treatment is of the very greatest importance. The heart failure is directly due to the arrhythmia, for the parts of the heart no longer work in that orderly manner designed by nature; consequently, treatment has a very definite aim before it. Further, the effect on these hearts of such a drug as digitalis is entirely different from the effect on a heart beating with a normal rhythm.

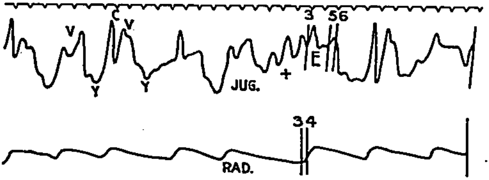

Fig. 24.—The jugular pulse is of the ventricular type and shows three small waves at + probably due to fibrillary contraction of the auricle.

I hope that I have been able to show how necessary it is for the proper appreciation of the functional pathology of the heart, to understand the meaning of the venous pulse, and also to show that a great field for research still lies before the student of clinical medicine.